Slowness and the preceding preparatory interval effect in schizophrenia.

By: Michael B. Miller, Loren J. Chapman, Jean P. Chapman and Thomas R. Kwapil

Miller, M.B., Chapman, L.J., Chapman, J.P., & Kwapil, T.R. (1993). Slowness and the preceding preparatory interval effect in schizophrenia. Journal of Abnormal Psychology, 102, 145-151. http://dx.doi.org/10.1037/0021-843X.102.1.145

Abstract:

Measured the reaction times (RTs) of 25 schizophrenic (SCZ), 69 matched normal, and 14 bipolar Ss to a tone preceded by a preparatory interval (PI) of varying length. RTs increase when the PI for the immediately preceding trial (PPI) is longer than the PI for the current trial. Several studies have shown that this PPI effect is heightened in schizophrenia. The authors replicated this finding. However, they found that the size of the PPI effect within groups increased with overall slowness and that the least squares regression line relating the PPI effect difference score to overall slowness did not differ between groups, nor did SCZ Ss' regression line differ from that of normal Ss. Group differences on the PPI effect were also analyzed by taking residuals for members of all groups from the normal Ss' regression line of the PPI effect difference score on overall slowness. Groups did not differ on these residuals, nor did SCZ Ss differ from normal Ss. The authors conclude that the heightening of the PPI effect in schizophrenia is like that observed in equally slow normal Ss. This finding suggests that the PPI effect does not appear to be a promising marker of a distinctive SCZ pathology. (PsycINFO Database Record (c) 2012 APA, all rights reserved)

Keywords: auditory stimulation | bipolar disorder | reaction time | schizophrenia | psychology

Article:

Acknowledgement: This research was supported by a Research Scientist Award MH00743 and by Research Grants MH44062 and MH31067 to Loren J. Chapman from the National Institute of Mental Health and by a grant from the Scottish Rite Schizophrenia Research Program to Loren J. Chapman.

We would like to thank Kathy Reilly and Shannon DeVries and the members and staff of the Off-the-Square Club for generously donating so much of their time and energy to this project.

Correspondence concerning this article should be addressed to: Michael B. Miller, Department of Psychology, University of Wisconsin—Madison, 1202 West Johnson Street, Madison, Wisconsin 53706

In studies of reaction time (RT) in which the preparatory interval (PI) between the warning signal and the imperative stimulus varies from trial to trial, the length of the PI for the immediately preceding trial (called the preceding preparatory interval, or PPI) affects the RT in the present trial. RTs are longer for most subjects when PPIs are longer than PIs (Niemi & Näätänen, 1981), but Zahn, Rosenthal, and Shakow (1963) found that this effect, often called the PPI effect, was more pronounced in schizophrenic subjects than in control subjects. Other investigators have replicated this finding (Kornetsky & Orzack, 1978; Nideffer, Neale, Kopfstein, & Cromwell, 1971; Zahn, 1970; Zahn & Rosenthal, 1965). The PPI effect was measured in all of those studies as the difference score of RT for trials on which the PPI was greater than the PI minus RT for trials in which PPI was less than or equal to the PI. We present data indicating that this PPI effect difference score is positively correlated with overall slowness within the normal population and that the PPI effect is heightened in schizophrenic subjects only as much as in equally slow normal subjects.

Interpretations of the Heightened Preceding Preparatory Interval Effect in Schizophrenia

Many theories have been proposed to explain the enhanced effect of the PPI on the RTs of schizophrenic individuals. Zahn et al. (1963), following Shakow's segmental set hypothesis (see Shakow, 1977), held that instead of establishing a generalized preparatory set based on the full sequence of trials, schizophrenics base their patterns of preparation narrowly on a more recent experience, mostly on the immediately preceding PI. Zubin (1975) suggested that schizophrenics have a longer neural persistence of stimuli, a phenomenon that facilitates performance for a trial that is similar to its preceding trial but inhibits performance for a trial that is dissimilar to its preceding trial. Thus, schizophrenics should be least impaired when the PPI is equal to the PI and most impaired when the PPI is much different from the PI. Zubin added that this effect should be greatest when a long PPI precedes a short PI because short PIs allow the least amount of time for the subject to readjust before responding. Spring and Zubin (1977) explained schizophrenics' heightened PPI effect as resulting from the influence of the PPI on the patients' estimates of the passage of time during the PI. When the PPI is long, schizophrenics judge the time passed in the PI to be briefer than it is and thus are unprepared for the imperative stimulus. Salzinger (1971) argued that schizophrenics' heightened PPI effect is caused by their behavior being primarily controlled by stimuli that are “immediate” in the environment, with one dimension of immediacy being recency of stimulus. Thus, schizophrenics tend to prepare for a PI of the same length as the

PPI, leaving them unprepared when the PI is shorter. Callaway and Naghdi (1982) accepted this immediacy explanation and further suggested that it reflects, in schizophrenia, a prolonged activation of neural activity that accompanies automatic parallel processing, which they assumed characterizes the PPI effect. Cromwell and Dokecki (1968) explained schizophrenics' heightened PPI effect by a failure to “disattend” (to withdraw attention) from the stimuli that occur immediately prior to the test stimulus. Broen (1968) similarly explained it by a restriction of memory scanning and of cue utilization to the strongest stimuli so that the PPI, being stronger because of its greater recency than other PIs, achieves undue influence on the subject's readiness to respond.

Nideffer et al. (1971) suggested that schizophrenics' heightened PPI effect may really be a heightened PI effect in disguise because short PIs, which yield the largest deficits in schizophrenics are also the PIs that are more often preceded by a longer PI. Nideffer et al. suggested that the heightened PPI effect in schizophrenia disappears when the PI is held constant. They found support for this hypothesis by comparing schizophrenics with a psychiatric control group. These groups, however, did not differ on overall slowness.

Prediction of the Preceding Preparatory Interval Effect From Overall Slowness

We propose that the heightened PPI effect in schizophrenia may be predictable from slowness of response. Overall slowness, in the sense of a slowing of all measured responses, is often expected to enlarge difference scores of slowness. For example, if a subject takes 25 ms longer for Response A than for Response B, a doubling of the RT of both responses will double the latency difference to 50 ms. As we report, the heightened PPI effect in schizophrenia can be described in terms of the normal relation of PPI effect to overall slowness, although our data cannot establish a causal relation between the two. This statement of a relation is not meant to imply that the RTs of schizophrenics are mere multiples of normal RTs but that schizophrenic PPI scores are like those of equally slow normal subjects. This distinction is important because slowness within the normal group does not appear to be controlled completely by variation of a single multiplicative factor. If schizophrenic subjects are like normal subjects but slower, one would expect the RTs of schizophrenics to be determined by a complex of factors, including but not limited to multiplicative slowing.

Zahn et al. (1963) recognized that the raw difference scores that measure the PPI effect might be inflated by schizophrenics' overall slowness. In order to rule out this possible artifact, Zahn et al. computed an additional analysis in which subgroups of schizophrenic and normal subjects selected to match on overall slowness were compared on the PPI effect. They selected fast schizophrenics and slow normals so as to match for mean latency on trials in which the PPI was less than or equal to the PI. They then compared these groups on the PPI effect difference score of mean latency when the PPI was greater than the PI minus mean latency when the PPI was less than or equal to the PI. The matched groups differed significantly, and Zahn et al. concluded that

overall latency is relatively unimportant in determining PPI effect differences between schizophrenic and normal populations.

However, the Zahn et al. (1963) finding of a difference between matched groups could have been caused by a regression artifact. Specifically, when subgroups selected from groups that differ on overall slowness are matched on one measure of slowness (e.g., latency when the PPI is less than or equal to the PI), the matched subgroups will tend to have scores closer to the means of their larger groups on any other measure of slowness (e.g., latency when the PPI is greater than the PI). This follows from the imperfect correlation of the two RT tasks.

Method

Subjects

Schizophrenic group

Twenty-five schizophrenic subjects (20 men and 5 women) were recruited through a social service agency that provides inexpensive meals for current and former psychiatric patients. All of the subjects in the study were paid for their participation. One additional female schizophrenic subject was dropped because she was unable to perform the task. All schizophrenic subjects met the criteria for chronic schizophrenia listed in the revised third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM–III–R; American Psychiatric Association, 1987). Diagnostic information was obtained using a modified form of the Schedule for Affective Disorders and Schizophrenia–Lifetime Version (Spitzer & Endicott, 1977) that contained portions of the Diagnostic Interview Schedule (Robins, Helzer, Croughan, & Ratcliff, 1981) and the Mini-Mental State Examination (Folstein, Folstein, & McHugh, 1975). Detailed psychiatric histories from hospital records were used for diagnosis, together with the interview material. Each diagnosis was discussed and unanimously agreed to by three of the authors. Twenty schizophrenics were in an active phase of psychosis at the time of testing and met the DSM–III–R criteria for the following subtypes: 10 paranoid, 4 disorganized, and 6 undifferentiated. Five schizophrenics met criteria for the residual subtype. Seventeen schizophrenics were receiving antipsychotic medication at the time of testing, a mean daily dose, expressed as chlorpromazine equivalents (Davis, 1985), of 632 mg. Eight were receiving no medication.

Normal control group

Sixty-nine normal control subjects (55 men and 14 women) were recruited through telephone solicitation of people listed in the Madison, Wisconsin, city directory. We obtained a relatively large normal control group in order to get a stable estimate of the regression of the PPI effect on overall slowness (see the Results section). Following the experiment, each normal subject was questioned about his or her personal and family history of psychiatric illness and substance abuse. Twenty-six additional subjects were excluded from the normal group: Twelve reported a personal history of psychiatric illness, 10 reported a history of substance dependence, and 4

reported having a first-degree relative with a history of psychiatric illness. This group was selected to match the schizophrenic group on mean and variance of years of age (schizophrenic subjects, M = 37.5, SD = 7.5; normal subjects, M = 37.1, SD = 6.1); highest social position attained by either parent as measured by Hollingshead's (1957) Two-Factor Index of Social Position (schizophrenic subjects M = 38.7, SD = 14.3; normal subjects M = 42.4, SD = 14.1); and gender (20% female and 80% male for both groups).

Bipolar group

Fourteen subjects (11 men and 3 women) meeting the DSM–III–R criteria for bipolar disorder (12 with a history of psychosis) were recruited and assessed in the same way as were the schizophrenic subjects. We wished to include a bipolar group because such subjects would often have been diagnosed schizophrenic in earlier RT studies. These subjects matched the schizophrenic and normal groups on gender and on mean and variance of age (M = 37.7, SD = 6.8). Eight bipolar subjects were receiving antipsychotic medication, a mean daily dose, expressed as chlorpromazine equivalents, of 297 mg. Five were receiving lithium carbonate, a mean daily dose of 1,660 mg, and 5 were receiving no medication.

Apparatus

A modified Compaq (Houston, TX) Portable Computer, Model 101709, was used to control the delivery of the imperative stimulus, the timing of the PI, and the recording of subjects' RTs. The imperative stimulus tone was produced by an Intersil (Cupertino, CA) Precision Waveform Generator (Model ICL8038), whose rise time was resistorcapacitor controlled to an exponential form. The imperative stimuli, the white noise, and the ready signals were presented through Koss (Milwaukee, WI) Model K-6 headphones. Background white noise was generated with a Grason-Stadler (West Concord, MA) Model 901B noise generator.

Procedure

Subjects wearing headphones, rested their index finger on a springloaded button. They were told to depress the button after the experimenter said “ready” and when they felt ready, and to keep the button depressed until the tone sounded, at which time they should release the button as quickly as possible. The imperative stimulus was a 1-s 1000-Hz tone of 70 dB with a rise time of 8 ms. There was also a constant background white noise of 55 dB.

Subjects' depression of the button initiated the PI and thus served as the warning signal. Five PIs—1, 2, 4, 7, and 15 s—were used, as in Experiment 2 by Zahn et al. (1963). The sequence was such that every PI followed every other PI, including itself, once and only once, in each series of 26 trials. There was approximately a 4.5-s interval between subjects' responses to the imperative stimulus and the experimenter's initiation of the next trial. Four 26-trial blocks were administered, twice as many as in the Zahn et al. Experiment 2, with 2-min rest periods following the first and third blocks, and a 5-min rest period following the second block. During

the 5-min break, the subjects rated the “funniness” of 20 cartoons on a 1–5 scale. The cartoon rating task was chosen because it provided a pleasant diversion and prevented subjects from discussing the experiment with the experimenter.

Normal subjects were not told that other groups were being studied, and no one was told the hypothesis about the effect of the length of the PPI on RT. After all RTs had been collected, normal subjects were asked about their family and personal histories of psychiatric illness and drug and alcohol abuse; the clinical subjects were given an extensive psychiatric interview during a separate session.

Results

In all of the following analyses, unless specified otherwise, trials were not scored if the subject either responded before the imperative signal or took more than 2,000 ms to respond. The following percentages of trials were not scored: schizophrenic, 1.64%; bipolar, 0.57%; and normal control, 0.12%. RT means were taken from the remaining trials; RT medians were based on all trials except for those on which subjects responded before the imperative tone.

Classical Measures of the Preceding Preparatory Interval Effect

We used the following standard analytical techniques, not because we preferred them but because we wished to determine whether we had replicated the findings of previous researchers that schizophrenics show larger PPI effect latency differences than do normal subjects. First, in an analysis modeled after one of Zahn et al. (1963), we defined the PPI effect as the difference score of the mean RT for trials in which the PPI was greater than the PI minus the mean RT for trials in which the PPI was less than or equal to the PI. The schizophrenic, bipolar, and normal control groups were compared in a Groups × Tasks repeated measures analysis of variance (ANOVA), with mean latency as the dependent variable. The main effects for groups and for tasks were both significant, $F(2, 105) = 20.20$, $p < .0001$, and $F(1, 105) = 82.70$, $p < .0001$, respectively, indicating that the groups differed in overall latency and that a significant PPI effect was present in the combined group of all subjects. The Groups × Tasks interaction was also significant, $F(2, 105) = 8.03$, $p < .001$, indicating that groups differed in the magnitude of the PPI effect. The schizophrenics showed a larger PPI effect than did their normal control group, Welch's $t(26) = 2.67$, $p = .02$, and the bipolar group did not differ from either the schizophrenic group Welch's $t(25) = 0.05$, or the normal group, Welch's $t(14) = 1.82$. This finding of a difference between schizophrenic and normal groups in the PPI effect replicates the finding of Zahn et al. (1963) in their Experiment 2 and is consistent with the findings of Kornetsky and Orzack (1978), Nideffer et al. (1971), Zahn (1970), and Zahn and Rosenthal (1965). The meaning of the finding is, however, ambiguous because the size of the PPI effect difference score may be in part an expression of overall slowness. We return to this relationship in later regression analyses.

Controlling for the confounding effect of the PI

We next tested the Nideffer et al. (1971) hypothesis that the heightening of PPI effect difference scores in schizophrenia is not an effect of the PPI per se and can be better understood as an effect of the PI because shorter PIs tend to yield a larger difference between schizophrenic and normal subjects. Because PIs of each length follow PIs of every length equally often, when the PPI is longer than the PI, PIs tend to be short; when the PPI is shorter than the PI, PIs tend to be long. Therefore, the PPI effect difference score used by Zahn et al. (1963), and in our study, measures both the PPI effect and the PI effect. Nideffer et al. hypothesized that schizophrenics' heightened PPI effect disappears when the PI is held constant. In order to test the Nideffer et al. hypothesis, we used a modified version of their procedure to measure the PPI effect with PI held constant. Median latencies were used. For every subject, the PPI effect difference score was taken for trials with a PI of 2 s, and a separate PPI effect difference score was taken for trials with a PI of 4 s. Trials with a PI of 1 s were omitted because there were few trials with a PPI shorter than or equal to 1 s, and trials with PIs of 7 or 15 s were omitted because there were few or no trials with a longer PPI. The PPI effect difference scores for 2-s and 4-s PIs were summed to produce a PPI effect measure that was not confounded with PI. Schizophrenic, bipolar, and normal control groups were compared in a one-way ANOVA on this sum. All groups showed highly significant PPI effects. Groups differed on the PPI effect, Welch's $F(2, 25) = 5.92$, $p < .01$, and schizophrenics showed a larger PPI effect than normal controls, Welch's $t(29) = 3.22$, $p < .01$. With their larger variance and smaller sample size, the bipolar group did not differ significantly from either of the other two groups, even though the bipolar group had the largest mean PPI effect score. Clearly, the heightened PPI effect in schizophrenia was not attributable to the PI effect. The discrepancy between our finding and that of Nideffer et al. (1971) might have been caused by their use of a psychiatric control group that did not differ from their schizophrenic group on overall slowness.

Comparison of groups matched on overall latency

In order to replicate an analysis of Zahn et al. (1963), we performed another analysis that we do not recommend. We selected subgroups of slow normal subjects and fast schizophrenic subjects so that subgroup means would be matched for mean latency on trials in which the PPI was less than or equal to the PI. Thirty-two normal subjects (M = 268 ms, SD = 32 ms) and 15 schizophrenic subjects (M = 271 ms, SD = 33 ms) were selected. The schizophrenic subgroup was found to exceed the matched normal subgroup on the mean PPI effect difference score, $t(45) = 2.93$, $p < .01$. This finding closely paralleled the finding of Zahn et al. The significant difference for these matched subgroups could have arisen from statistical regression.

Comparisons Based on Regression of Difference Scores on Overall Latency and Lord's Paradox

The analyses presented thus far were for the purpose of showing that our results essentially replicate those of Zahn et al. (1963) when the same modes of analysis are used. We have argued that these modes of analysis are flawed. We now present regression analyses that we believe are more appropriate.

One cannot determine whether normal and schizophrenic subjects would differ on the PPI effect difference score if they did not differ on overall latency. Lord (1967) pointed out that when naturally occurring groups differ on the covariate, the use of an analysis of covariance (ANCOVA) and other methods of making allowances for group differences on the covariate in evaluating group differences on the criterion yield contradictory conclusions that he termed a "paradox." Lord concluded that all such attempts are invalid and that statistical methods cannot answer this kind of question. Other writers have recapitulated and elaborated on this conclusion (Cronbach & Furby, 1970; Holland & Rubin, 1983; Huitema, 1980; Kenny, 1979). Because the structure of the causal system relating the criterion variable to the covariate is unknown in each group, controlling for the covariate cannot indicate whether the groups would differ on the criterion variable if they did not differ on the covariate. One cannot determine by statistical methods what the state of nature would be if it were different than it is. Holland and Rubin (1983) pointed out, however, that one can nevertheless use an ANCOVA validly to make useful descriptive statements in situations in which causal conclusions would not be justified. This view is consistent with Lord's (1963) own example of a suitable ANCOVA. We follow the lead of Holland and Rubin and of Lord in the analyses that follow.

We conducted a series of regression analyses in order to determine whether the schizophrenics would be more deviant on the PPI effect than the normal subjects of similar overall latency. In the first of these analyses, we compared regression lines between groups to determine whether group membership would result in different relations between the PPI effect difference score and overall latency. In the second group of regression analyses, for every subject in all three groups, we obtained residual scores from the normal subjects' regression line of the PPI effect difference score on overall slowness. This is an unusual adaptation of the regression method. Analysis of such residual scores reveals whether individuals or groups of subjects have larger or smaller PPI effect difference scores than expected by normal standards. Note that this is not a test of whether normal and schizophrenic differences on the PPI effect are caused by differences in overall latency.

We used medians rather than means in all of these analyses because medians are affected less by occasional atypically long latencies. The least squares line for the regression of the PPI effect difference score—the median latency when the PPI was greater than the PI minus the median latency when the PPI was less than or equal to the PI—on overall slowness (the sum of the same two median latency scores) was computed for each of the three groups of subjects.

Leverage of deviant cases

In order to avoid making biased estimates of regression parameters as a result of including deviant observations, we obtained the leverage of each case within each of the three groups and used it to eliminate deviant cases. Leverage is a case statistic that conveys the potential for influence of a single case on the regression (see Hamilton, 1992, for a nonmathematical discussion). This statistic is based only on the predictor scores, not on the response scores. In the

present study, subjects with high leverage were so deviantly slow that they were in a position to exert undue influence on the estimation of the slope and intercept of the regression line. Because we wanted to avoid such an outcome, measurement of leverage appeared to be appropriate. In straight-line regression, the leverage of the ith case is $L_i = (1 + z_i^2) \div n$, where z_i is the standardized score (m = 0, variance = 1) on the predictor variable for the ith subject in a group of size n. The sum of subjects' leverages within the group for which the regression is computed is equal to 2.0, so that the average leverage of a case within the group (of size n) is equal to $2/n$. According to Huber (1981), cases with leverages greater than 0.5 should be avoided when possible. The maximum leverage within the normal control group was only 0.17, but the maximum leverage within the schizophrenic group was 0.68, and within the bipolar group it was 0.73. These findings indicate that one extraordinarily slow subject in each of the two clinical groups exerted undue control over the regression for their group. The schizophrenic subject was 724 ms slower on the sum of median latencies than the next slowest schizophrenic subject and was 1, 172 ms from the schizophrenic group median on the same measure. The bipolar subject was 456 ms slower on the sum of median latencies than the next slowest bipolar subject and was 687 ms from the bipolar group median on the same measure. The single schizophrenic leverage case influenced the regression massively within the schizophrenic group: With the case included, the estimated slope of the regression of difference on sum was 0.03, but with the case excluded the estimated slope was 0.15, an increase of more than 4 SE units of the estimated slope for the full schizophrenic sample. We excluded the 1 schizophrenic leverage case and the 1 bipolar leverage case for the comparison of groups' regression lines. In the groups that remained, maximum leverage was 0.33 in the schizophrenic group and 0.34 in the bipolar group.

Comparison of regression lines

With leverage cases dropped, we compared the regression lines for schizophrenic, bipolar, and normal control groups using the nested procedure recommended by Seber (1977, pp. 200–201), in which groups' regression lines are tested first for parallelism and then, if parallelism cannot be rejected, they are tested for equal intercepts assuming parallelism; if neither test is significant, the overall test of coincident (identical) regression lines is then conducted. In all three of these tests, the residual sums of squares from the two models being compared are combined with the degrees of freedom to produce an F statistic. Groups did not differ on regression slopes, $F(2, 100) = 0.41$, on intercepts given equal slopes, $F(2, 102) = 0.32$, or on the regression line as a whole, $F(4, 100) = 0.37$. The regressions for each group appeared to be linear; the parameters of the three regression lines are shown in Table 1. In a second analysis, excluding the bipolar group, schizophrenic and normal control groups did not differ on regression slopes, $F(1, 89) = 0.99$, on intercepts given equal slopes, $F(1, 90) = 0.55$, or on the regression line as a whole, $F(2, 89) = 0.77$. This lack of difference between the regression lines for normal and schizophrenic subjects meant that at any given level of slowness, normal and schizophrenic subjects showed similar PPI effect difference scores. The degree of similarity between schizophrenic and normal subjects on this relation of PPI score to slowness can be clarified by considering how much of the variance

of the PPI effect difference scores can be accounted for by a single regression line of PPI effect on slowness and how much additional variance is accounted for by the use of two regression lines, one for each group. For the schizophrenic and normal groups, with two separate lines, 54.1% of the variance in the PPI effect could be accounted for by overall slowness; with a single line, 53.3% was accounted for. In other words, in the prediction of the PPI effect difference score, the use of group information (schizophrenic vs. normal control) added only a statistically insignificant increment of 0.8% to the predictive validity obtained through the use of overall slowness alone. A comparison of normal and bipolar groups revealed a similar lack of difference of regression lines, $F(2, 78) = 0.63$. These findings indicate that PPI effect difference scores of schizophrenic and bipolar subjects were like those of normal subjects at the same level of overall slowness.

Table 1
Regression Coefficients and Pearson Product–Moment Correlations for the Prediction of the Preceding Preparatory Interval Effect Difference Score (A − B) From Overall Slowness (A + B)

Group	*n*	Slope	Intercept (in ms)	*r*
Normal control	69	0.12	−40	.54*
Schizophrenic	24	0.15	−56	.69*
Bipolar	13	0.15	−53	.48
Normal (Zahn, Rosenthal, & Shakow, 1963)	9	0.12	−29	.46

Note. Slopes are unit free (ms/ms). The regression estimates for the schizophrenic and bipolar groups were computed after dropping the 1 deviant subject from each group, as described in the text. Sample sizes (*n*) are those for regression computations.
* $p < .001$.

When the bipolar leverage case was included, the results were unchanged, but when the schizophrenic leverage case was included, the regression within the schizophrenic group changed so sharply that the hypothesis of equal within-groups regressions across the three groups was rejected at the .001 level, $F(4, 102) = 6.67$. We report this for completeness, but the validity of the test is highly questionable given the massive influence of the single schizophrenic outlier.

Comparison of groups on residuals from the normal subjects' regression line

An alternative method of analyzing these data was to compute the regression of the PPI effect difference score on the measure of overall slowness for normal subjects and to use this regression line to calculate a residual PPI effect score for every subject in the bipolar, schizophrenic, and normal groups. The groups could then be compared on these residualized scores as a measure of the extent to which their PPI scores depart from those expected, by the standards of normal subjects, given their scores of overall slowness.

The PPI effect difference score and the measure of overall slowness were defined as in the previous analyses. We excluded the 1 schizophrenic leverage case and the 1 bipolar leverage case because their scores of overall slowness were so far outside of the range of all other subjects

on overall slowness that their residual scores were of questionable validity. Groups did not differ on the PPI effect residuals, Welch's $F(2, 24) = 1.43$; schizophrenic subjects did not differ from normal subjects, Welch's $t(27) = 1.37$; and bipolar subjects did not differ from normal subjects, Welch's $t(12) = 0.70$. Repeating the analysis with the schizophrenic outlier included, groups did not differ on PPI score residuals, Welch's $F(2, 24) = 0.60$; schizophrenic subjects did not differ from normal subjects, Welch's $t(26) = 0.15$; and schizophrenics' mean residuals did not differ from zero, $t(24) = 0.15$. As an alternative to a t test, and retaining the outlier, we performed a Wilcoxon rank-sum test comparing schizophrenic and normal groups on the residuals. This test, being based on ranks, is not sensitive to the score of the schizophrenic outlier. Again, schizophrenic and normal groups did not differ ($z = 0.82$). These analyses indicated that the mean PPI effect difference scores for schizophrenic and bipolar groups could be predicted from their overall slowness using the regression line from the normal group.

Comparison of Zahn et al.'s (1963) groups on residuals from his normal subjects' regression line

We repeated this analysis of residualized PPI scores using the data from Experiment 2 of Zahn et al. (1963) in order to determine whether their findings differed from ours. We first calculated the slope of the regression of the PPI effect difference score on the latency sum for the Zahn et al. normal subjects using the standard deviations given in their Table 2. The calculation makes use of the covariance that can be obtained from the tabled standard deviation of the difference score and the fact that the slope of the difference score on the sum is given by the difference of the variances divided by the variance of the sum (see Chapman & Chapman, 1988). This slope was approximately 0.12, virtually identical to the slope for our normal group (see Table 1). On the other hand, because Zahn et al. used only 9 normal control subjects, the standard error of their regression slope was large (also 0.12) so that the degree of similarity of their finding with our own must be viewed as a coincidence.

Zahn et al. (1963) did not compare groups on the residualized difference scores that would be yielded from the regression of the PPI effect difference score on overall slowness. One can, however, duplicate the results of such an analysis using the means and standard deviations given in the Zahn et al. (1963) Table 2 and relying on the following algebraic relationships.

Consider the linear regression equation for predicting the raw difference score from overall slowness,

$$(A - B) = \beta(A + B) + \alpha$$

where (A – B) represents the predicted PPI effect difference score, (A + B) represents the observed overall slowness score, and β and α are the slope (raw regression coefficient) and intercept estimated using data from a large normal sample. The residual

$$\begin{aligned}(A - B) - (A - B) &= (A - B) - \beta(A + B) - \alpha \\ &= (1 - \beta)A - (1 + \beta)B - \alpha,\end{aligned}$$

where (A – B) represents the observed PPI effect difference score. The α is simply a constant subtracted from every subject's score, so one can ignore it and compare subjects and groups on (1 − β) A – (1 + β) B. In the present study, β = 0.12, so in comparing groups on residualized scores, we have effectively compared groups on (0.88 A – 1.12 B). Conceptualizing residualized scores in this way allowed us to perform a t test, using Zahn et al. (1963, Table 2), comparing schizophrenic and normal groups on residualized scores. 1 Groups did not differ, Welch's $t(28) = 0.61$. In addition, the mean residual for the Zahn et al. schizophrenics did not differ significantly from zero, $t(20) = 0.71$. These results are highly similar to those from our own data.

Discussion

Our finding that the regression line relating the PPI effect difference score to overall slowness did not differ between schizophrenic and normal groups indicates that the normal subjects' relationship between slowness and the PPI effect was present in a closely similar form in schizophrenia. This finding, combined with the finding that our outpatient schizophrenic subjects did not differ from normal control subjects in their PPI effect residuals from the normal subjects' regression line, indicates that the schizophrenics' raw difference scores were similar to those of equally slow normal subjects. Of course, it is possible that more disturbed schizophrenics would show a greater PPI effect than expected from their slowness, but a lack of severity of illness did not appear to account for the difference between our findings and those of Zahn et al. (1963), who tested inpatients. In fact, our findings paralleled those of Zahn et al. (1963) to a remarkable degree when comparable modes of analysis were used. We included bipolar subjects because the Zahn et al. schizophrenics, being diagnosed by the criteria of a predecessor of the DSM–III, might have included some patients who would now be labeled bipolar. Although our bipolar group was small, the data did not indicate that bipolar subjects had a heightening of the PPI effect relative to schizophrenics. Therefore, even if some of the Zahn et al. schizophrenics were to be deleted because they would be diagnosed as bipolar under the DSM–III–R, the results of their group comparisons would probably not be altered substantially.

It is interesting to compare our finding with that of Rosenthal, Lawlor, Zahn, and Shakow (1960), who found that Rodnick and Shakow's (1940) “set index,” which was derived to maximally discriminate schizophrenic from normal subjects using serial RT data, had a high rank order correlation (.92) with mean RT within the schizophrenic group. We propose, in effect, that a second schizophrenic RT defect—the PPI effect—can also be predicted from RT.

We chose to measure overall slowness by the sum of two median latency scores. This choice was not arbitrary, although one might reasonably argue for other slowness measures. For example, when considering an (A – B) difference score, one might use B as a measure of slowness. A problem with this approach is that errors of measurement of B contribute a negative component to the correlation of (A – B) with B. This will tend to reduce the correlation to an extent determined by the error variance of B. On the other hand, errors of measurement in both A and B affect the correlation of (A – B) with (A + B), but in opposite directions. Given similar

reliabilities for A and B, the effects of error will tend to cancel. A second reason for using (A + B) is that it is used in the usual comparison of groups on overall latency in a Groups × Tasks repeated measures ANOVA, using latency on each task as the dependent measure. That is, the test of the main effect for groups in the repeated measures ANOVA is identical to a one-way ANOVA comparing group means on (A + B) latency sums. Thus, the (A + B) sum has been used implicitly as a measure of overall slowness by many researchers in their ANOVAs. A third reason for using (A + B) is that it is a more reliable measure than B alone. Ultimately, it would probably be better to design studies so that an independent measure of overall slowness is available (i.e., a measure that theoretically does not share error variance with the PPI effect difference score), but the design of our study was based on an earlier study that included no such independent measure.

Lord's Paradox Revisited

The conclusion we wish to draw from the comparison of group regression lines and residualized difference scores is descriptive, not causal. That is, the expected value of the difference score is nearly equal for schizophrenic and normal subjects of the same overall latency. This does not logically imply that group differences on the difference score are caused by group differences in overall latency. Such an inference could be derived only if it were safe to assume that individual differences in difference scores were caused only by individual differences in slowness (plus error). This assumption cannot be tested. Therefore, we do not attempt to draw a causal conclusion.

Conclusion

The scores of the PPI effect of schizophrenics were like those of normal subjects who were equally slow. A behavior that serves as a marker of schizophrenic pathology is usually expected to be much more deviant in schizophrenic subjects than in normal subjects with slow RTs. Therefore, the PPI effect does not appear promising as such a marker.

Footnotes

1 A document fully spelling out these algebraic relationships is available from Michael B. Miller.

References:

American Psychiatric Association. (1987). Diagnostic and statistical manual of mental disorders (rev.) (3rd ed.). Washington, DC: Author.

Broen, W. E. (1968). Schizophrenia: Research and theory. San Diego, CA: Academic Press.

Callaway, E., & Naghdi, S. (1982). An information processing model for schizophrenia. Archives of General Psychiatry, 39, 339– 347.

Chapman, L. J., & Chapman, J. P. (1988). Artifactual and genuine relationships of lateral difference scores to overall accuracy in studies of laterality. Psychological Bulletin, 104, 127– 136.

Cromwell, R. L., & Dokecki, P. R. (1968). Schizophrenic language: A disattention interpretation. In S.Rosenberg & J. H.Koplin (Eds.) , Developments in applied psycholinguistics research (pp. 209– 261). New York: Macmillan.

Cronbach, L. J., & Furby, L. (1970). How we should measure “change”—Or should we?Psychological Bulletin, 74, 68– 80.

Davis, J. M. (1985). Antipsychotic drugs. In H. I.Kaplan & B. J.Sadock (Eds.) Comprehensive textbook of psychiatry (4th ed., (pp. 1481– 1512). Baltimore, MD: Williams & Wilkins.

Folstein, M. F., Folstein, S. E., & McHugh, P. R. (1975). Mini-Mental State: A practical method for grading the cognitive state of patients for the clinician. Journal of Psychiatric Research, 12, 189– 198.

Hamilton, L. C. (1992). Regression with graphics: A second course in applied statistics. Pacific Grove, CA: Brooks/Cole.

Holland, P. W., & Rubin, D. B. (1983). On Lord's paradox. In H.Wainer & S.Messick (Eds.) , Principals of modern psychological measurement: A festschrift for Frederic M. Lord (pp. 3– 25). Hillsdale, NJ: Erlbaum.

Hollingshead, A. B. (1957). Two-Factor Index of Social Position Unpublished manuscript, Yale University, New Haven, CT.

Huber, P. J. (1981). Robust statistics. New York: Wiley.

Huitema, B. E. (1980). The analysis of covariance and alternatives. New York: Wiley Interscience.

Kenny, D. A. (1979). Correlation and causality. New York: Wiley Interscience.

Kornetsky, C., & Orzack, M. H. (1978). Physiological and behavioral correlates of attention dysfunction in schizophrenic patients. Journal of Psychiatric Research, 14, 69– 79.

Lord, F. M. (1963). Elementary models for measuring change. In C. W.Harris (Ed.) , Problems in measuring change (pp. 21– 38). Madison: University of Wisconsin Press.

Lord, F. M. (1967). A paradox in the interpretation of group comparisons. Psychological Bulletin, 68, 304– 305.

Nideffer, R. M., Neale, J. M., Kopfstein, J. H., & Cromwell, R. L. (1971). The effect of previous preparatory intervals upon anticipatory responses in the reaction time of schizophrenic and nonschizophrenic patients. Journal of Nervous and Mental Disease, 153, 360– 365.

Niemi, P., & Näätänen, R. (1981). Foreperiod and simple reaction time. Psychological Bulletin, 89, 133– 162.

Robins, L. N., Helzer, J. E., Croughan, J., & Ratcliff, K. S. (1981). National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics, and validity. Archives of General Psychiatry, 38, 381– 389.

Rodnick, E. H., & Shakow, D. (1940). Set in the schizophrenic as measured by a composite reaction time index. American Journal of Psychiatry, 97, 214– 225.

Rosenthal, D., Lawlor, W. G., Zahn, T. P., & Shakow, D. (1960). The relationship of some aspects of mental set to degree of schizophrenic disorganization. Journal of Personality, 28, 26– 38.

Salzinger, K. (1971). The immediacy hypothesis of schizophrenia. In H. M.Yaker, H.Osmond, & F.Cheek (Eds.) , The future of time: Man's temporal environment (pp. 272– 292). Garden City, NY: Doubleday.

Seber, G. A. F. (1977). Linear regression analysis. New York: Wiley.

Shakow, D. (1977). Segmental set: The adaptive process in schizophrenia. American Psychologist, 32, 129– 139.

Spitzer, R. L., & Endicott, J. (1977). Schedule for Affective Disorders and Schizophrenia– Lifetime Version (SADS-L). New York: New York State Psychiatric Institute.

Spring, B., & Zubin, J. (1977). Reaction time and attention in schizophrenia: A comment on K. H. Nuechterlein's critical evaluation of the data and theories. Schizophrenia Bulletin, 3, 437– 444.

Zahn, T. P. (1970). Effects of reduction in uncertainty on reaction time in schizophrenic and normal subjects. Journal of Experimental Research in Personality, 4, 135– 143.

Zahn, T. P., & Rosenthal, D. (1965). Preparatory set in schizophrenia. Journal of Nervous and Mental Disease, 141, 352– 358.

Zahn, T. P., Rosenthal, D., & Shakow, D. (1963). Effects of irregular preparatory intervals on reaction time in schizophrenia. Journal of Abnormal and Social Psychology, 67, 44– 52.

Zubin, J. (1975). Problem of attention in schizophrenia. In M. L.Kietzman, S.Sutton, & J.Zubin (Eds.) , Experimental approaches to psychopathology (pp. 139– 166). San Diego, CA: Academic Press.